CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 34-2022: A 57-Year-Old Woman with Covid-19 and Delusions

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PRESENTATION OF CASE

Dr. Michael D. Kritzer: A 57-year-old woman with major depressive disorder and coronavirus disease 2019 (Covid-19) was evaluated at a hospital affiliated with this hospital because she was having delusions that she was dead.

The patient had been in her usual state of health until 2 weeks before this presentation, when myalgias, cough, sore throat, nausea, and vomiting developed. She sought evaluation at the primary care clinic of an academic medical center affiliated with this hospital (the two hospitals are part of the same health care system). Nucleic acid testing of a nasopharyngeal swab was positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) RNA, and the patient was instructed to quarantine at home. She lived with her father and assisted him with activities of daily living; he also received a diagnosis of Covid-19.

During the following week, the patient's cough persisted, and new shortness of breath developed. Her symptoms worsened; she felt that she was unable to take care of her father. Emergency medical services were called, and the patient and her father were taken to the emergency department of the other hospital, where they were both admitted for worsening Covid-19 pneumonia. The patient was treated with supplemental oxygen, remdesivir, and dexamethasone. Treatment with remdesivir was stopped on hospital day 4 when the blood aminotransferase levels increased to three times the upper limit of the normal range.

During the hospitalization, the patient was noted to have intermittent anxiety, particularly when discharge planning for her father was discussed. She and her brother declined to have their father discharged to a rehabilitation center and instead planned for him to eventually return home to quarantine with the patient. On hospital day 6, the patient's oxygen saturation was normal while she was breathing ambient air, and the blood aminotransferase levels had improved. She was discharged home with instructions to quarantine and continue taking dexamethasone.

One day after discharge, the patient's brother spoke to her on the telephone.

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He thought that she seemed to be confused and unable to take care of herself, and he asked her to return to the emergency department of the other hospital.

On evaluation in the emergency department, the patient explained that she was unsure why her brother had asked her to return to the hospital, and she said that she wanted to go home. She also expressed that she felt anxious about being home alone after discharge and overwhelmed about needing to care for her father at home once he was discharged from the hospital. The myalgias, cough, and shortness of breath had abated; she had no fevers, visual or auditory hallucinations, or suicidal or homicidal ideation.

The patient had a history of major depressive disorder, which had been diagnosed during the second decade of life. At the time of diagnosis, she had been admitted to a psychiatric hospital and had received electroconvulsive therapy; thereafter, she had been discharged to a partial hospital program. She had been hospitalized for psychiatric symptoms twice since then, once for major depressive disorder and once for a mixed bipolar episode that was due to insomnia and anxious distress. The latter episode was associated with catatonic features and was treated with electroconvulsive therapy.

The patient had no history of suicidal or homicidal ideation or attempts and no history of violence. She had hypertension, diabetes, obesity, and gastroesophageal reflux disease. Medications included dexamethasone, bupropion, fluoxetine, olanzapine, losartan, metformin, and pantoprazole. Sulfa drugs had caused angioedema, and lisinopril had caused cough. The patient was born in the Caribbean and had emigrated four decades earlier, first to southwestern Europe and then to the United States 2 years later. She lived in an apartment in an urban area of New England with her father, who had mild dementia. She did not drink alcohol, smoke cigarettes, or use illicit substances.

On examination, the temperature was 37.2°C, the pulse 97 beats per minute, the blood pressure 153/95 mm Hg, the respiratory rate 20 breaths per minute, and the oxygen saturation 93% while the patient was breathing ambient air. The patient was alert and oriented but guarded, with a flat affect. She appeared to be more anxious than she had been during the previous hospitalization. She paced around the room and perseverated about the care of her father. The remainder of the examination was normal.

The blood levels of electrolytes and glucose were normal, as were the results of liver-function and kidney-function tests. The white-cell count was 11,490 per microliter (reference range, 4000 to 11,000), with neutrophil predominance; the complete blood count with differential count was otherwise normal. Urinalysis and a radiograph of the chest were normal. Treatment with dexamethasone was stopped, and the patient was admitted to the hospital to facilitate discharge to a rehabilitation center for continued care.

On hospital day 3, the patient was noted to be more withdrawn, and she began responding to questions with one-word answers or silence. When she was encouraged to speak more, she continued to perseverate about the care of her father. When she was asked to elaborate on her concerns, she stated, "He is dead. I am dead."

The patient appeared disheveled, sullen, and anxious. She laid in bed motionless with her eyes open and looking forward, and she responded briefly to questions in a quiet voice with slowed speech. Her thoughts were perseverative and tangential. There was no evidence that she had loosening of associations, hallucinations, or suicidal or homicidal ideation. She had poor insight and judgment. Memory, attention, concentration, abstract reasoning, and fund of knowledge were normal. When her arms and legs were lifted against gravity and released, they fell to the bed without resistance; with encouragement, she was able to move them. Muscle tone was normal, with no rigidity or waxy flexibility. Imaging studies were obtained.

Dr. Aaron B. Paul: Computed tomography (CT) of the head (Fig. 1), performed without the administration of intravenous contrast material, revealed no evidence of an acute territorial infarct, intracranial mass, or hemorrhage. There was nonspecific moderate confluent hypoattenuation involving the supratentorial white matter.

Dr. Kritzer: Clonazepam was administered, and the dose of olanzapine was increased. Admission to an inpatient psychiatric unit was recommended. During the next week, while awaiting placement in an inpatient psychiatric unit, the

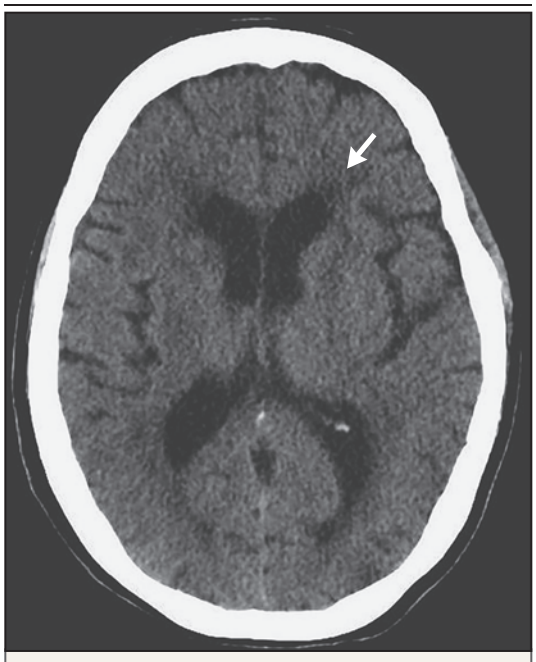

Figure 1. CT of the Head.
An axial image, obtained without the administration of intravenous contrast material, shows no evidence of an acute territorial infarct, intracranial mass, or hemorrhage. There is moderate confluent hypoattenuation involving the supratentorial white matter (arrow).

patient continued to show signs of anxiety and a depressed mood. She said, "I am dead. I do not exist. I am not real." She also believed that her father and brother, as well as her nurses and doctors, were dead. The patient was selectively mute and motionless, but she talked and moved with encouragement. She expressed that she felt directly responsible for the Covid-19 pandemic and asked to be thrown out of the window. She had the sensation that her bladder was gone and that she could not urinate, although she had been observed urinating independently. She felt that she could not eat, although she had been observed eating breakfast daily. On hospital day 9, the patient was transferred to the inpatient psychiatric unit of the other hospital.

A diagnosis and management decisions were made.

DIFFERENTIAL DIAGNOSIS

Dr. Gregory L. Fricchione: In this 57-year-old woman with metabolic syndrome and a mixed affective disorder suggestive of bipolar disorder, neuropsychiatric symptoms developed 2 weeks after the onset of Covid-19. The patient had psychomotor agitation, a flat affect, and anxious perseveration that was focused on her father's care. Three days later, she was noted to become motionless and hypophonic, with staring, speech latency, and verbal perseveration. Cognition was intact, but insight and judgment were impaired. In an attempt to explain her neuropsychiatric symptoms, I will consider the potential effects of medications that she had received, her underlying psychiatric disease, and her recent infection.

MEDICATION EFFECTS

This patient was receiving several medications for the treatment of a mixed affective disorder, including bupropion, fluoxetine, and olanzapine. Antidepressant medications could trigger a secondary manic episode, especially in this patient with suspected bipolar disorder. However, her presentation was not typical of drug-related mania, which has classic symptoms of insomnia, euphoria or irritability, extreme hyperactivity, and pressured speech. Although she was receiving psychotropic medications that have been associated with serotonin syndrome, there were no findings suggestive of this diagnosis, such as clonus, tremor, ataxia, hyperreflexia, or fever.[1]

This patient had recently started taking dexamethasone for the treatment of Covid-19. Glucocorticoids, particularly when administered at high doses, are potential triggers of a manic response commonly referred to as "steroid-induced psychosis." The use of glucocorticoids can cause a myriad of neuropsychiatric affective, cognitive, and behavioral symptoms.[2] The persistence of this patient's psychotic symptoms after the discontinuation of dexamethasone argues against the diagnosis of glucocorticoid-associated psychosis, although it is possible that dexamethasone triggered an underlying primary psychiatric disorder.

SEIZURES

The patient was noted to appear withdrawn, and at times, she would lie motionless and not respond to questions. These episodes suggest the possibility of complex partial seizures. Status epilepticus, including nonconvulsive status epi-

lepticus, has been reported in patients with Covid-19.[3] In addition, the patient was taking bupropion, a medication that has been associated with lowering of the seizure threshold. However, if her diminished responsiveness were due to nonconvulsive status epilepticus, I would expect her to have phases of deeper unresponsiveness fluctuating with brief phases of alertness with confusion. Because complex partial seizure disorder is often difficult to diagnose, I would perform long-term electroencephalographic (EEG) monitoring, while considering alternative diagnoses.

AUTOIMMUNE ENCEPHALITIS

Could this patient have autoimmune limbic encephalitis? The neuropsychiatric symptoms appear to have had a subacute onset followed by rapid progression, which suggests involvement of the limbic system. In addition, the white-matter changes observed on CT of the head suggest bilateral brain abnormalities. However, if the patient had autoimmune limbic encephalitis, I would expect white-matter changes to be restricted to the medial temporal lobes and an EEG to show focal temporal slowing.[4] I would perform magnetic resonance imaging (MRI) of the head and a lumbar puncture for cerebrospinal fluid (CSF) analysis to help rule out the diagnosis of autoimmune encephalitis, especially given the potential association of this condition with Covid-19.[5,6] Encephalitis associated with anti–*N*-methyl-D-aspartate (NMDA) receptor antibodies can lead to a neuropsychiatric presentation that often includes catatonic withdrawal, and it has been associated with viral illnesses.[7] A connection between encephalitis associated with anti–NMDA receptor antibodies and SARS-CoV-2 has not yet been established, but a potential relationship has been suggested.[8]

NEUROPSYCHIATRIC SYMPTOMS ASSOCIATED WITH COVID-19

Could this patient's neuropsychiatric symptoms be related to her recent diagnosis of Covid-19? Early studies suggested that more than one third of patients with Covid-19 had a neuropsychiatric syndrome.[9]

Some cases of Covid-19 lead to persistent symptoms or long-term complications that extend beyond acute disease (a condition sometimes referred to as postacute syndrome of Covid-19 or "long Covid").[10] In such cases, neuropsychiatric symptoms can include fatigue, myalgias, headache, anxiety, depression, dysautonomia, and cognitive impairment (also referred to as "brain fog").

In one study involving more than 60,000 patients with Covid-19, 18% of the patients had received a psychiatric diagnosis in the 14 to 90 days after infection.[11] Neuroinflammation is thought to play a role in Covid-19–related neuropsychiatric disorders,[12,13] and persistent autoantibodies have been detected in the CSF of patients with these conditions.[13-15]

New-onset psychosis has been reported in patients with Covid-19. In one report describing 10 patients, psychotic symptoms developed at least 2 weeks after the onset of Covid-19 symptoms, and structured delusions were common.[16] A recent systematic review of Covid-19–related psychosis cases confirmed that delusions were the most commonly reported psychotic symptom.[17] Of note, the majority of patients with Covid-19–related psychosis had only mild acute Covid-19 symptoms.

CT of the head performed in this patient revealed subcortical white-matter disease. The most frequent neuroimaging abnormalities observed in patients with Covid-19 involve white-matter changes.[18] Covid-19 has been associated with several white-matter diseases, including Covid-19–related disseminated leukoencephalopathy and cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL).[19,20] However, in this patient, there was no report of features that would be suggestive of these diagnoses, such as a clinically significant reduction in the level of consciousness, headaches, cranial nerve signs, sensorimotor deficits, gait defects, or changes in the deep-tendon reflexes. In addition, there were no other CT findings, such as microhemorrhages or lacunar infarcts. MRI of the head would be the next step to help rule out neuropsychiatric complications of Covid-19.

CATATONIA

This patient had several features suggestive of catatonia. If the Bush–Francis Catatonia Rating Scale were used, this patient would acquire points for mutism, withdrawal, immobility and

Table 1. DSM-5 Criteria for Catatonia Associated with a Major Mood Disorder and a General Medical Condition.*

≥3 of the following:
Catalepsy
Waxy flexibility
Stupor†
Agitation
Mutism†
Negativism†
Posturing
Mannerisms†
Stereotypies
Grimacing
Echolalia
Echopraxia

* DSM-5 denotes *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition.
† These criteria were present in the patient on hospital day 3.

stupor, staring, verbal perseveration, and autonomic instability, with a score of approximately 13 (on a scale ranging from 0 to 23, with higher scores indicating more severe catatonia).[21] On the basis of these reported findings on examination, the patient would meet the criteria in the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5), for catatonia associated with a major mood disorder and a general medical condition (Table 1).[22]

Catatonia is a disorder of the cingulate cortico–striato–thalamo–cortical circuits that results in the disconnection of motivation and movement, and it has multiple neuromedical and psychiatric causes.[23,24] Catatonia has been reported in several patients with Covid-19.[25] In a small study that evaluated the results of positron-emission tomography and CT of the head performed in patients with Covid-19 encephalopathy, there was evidence of persistent hypometabolism in the prefrontal cortex, anterior cingulate cortex, insula, and caudate cortico–striato–thalamo–cortical network.[26]

It is possible that this patient had Covid-19–related changes in the blood–brain barrier and choroid plexus that disrupted the cingulate cortico–striato–thalamo–cortical circuits and increased her risk of catatonia. Neuroleptic-induced catatonia related to the use of olanzapine is another possibility. In addition, the patient had a history of hospitalization for probable bipolar affective psychosis and catatonia, and bipolar disorder is the most common cause of psychogenic catatonia. There was no history suggestive of catatonia caused by conversion disorder.

The patient's catatonic symptoms abated after treatment with a benzodiazepine, which is the first-line treatment for catatonia. However, one of the most striking features of her presentation remains to be explained: her persistent thoughts that she was dead.

COTARD'S SYNDROME

This patient expressed self-deprecation and guilt about not being able to care for her father, and she had mood-congruent delusions that she and others were dead, along with a delusion that her bladder had disappeared. Her presentation is consistent with Cotard's syndrome, a syndrome included in the DSM-5 category of delusional misidentification syndromes.[27,28] Patients with Cotard's syndrome have nihilistic delusions, such as the belief that they are dead, have lost their souls, or are rotting inside, without functional organs or limbs. Three subtypes of Cotard's syndrome have been described: psychotic depression (a disorder associated with melancholia and nihilistic delusions), type 1 (a nondepressive delusional disorder), and type 2 (a disorder associated with mixed symptoms, including anxiety, depression, and auditory hallucinations).[29] Cotard's syndrome has been reported in at least one patient with Covid-19,[30] and catatonia and Cotard's syndrome may occur concurrently.[31,32]

Support and reassurance are key in the treatment of patients with Cotard's syndrome, but trying to talk patients out of their delusions is futile. Successful treatment of the underlying condition often helps the delusions to recede, although the delusions may wax and wane in patients with persistent depression and may become chronic in patients with schizophrenia. Multiple antipsychotic medications have been reported to reduce the symptoms of Cotard's syndrome. If medications fail, electroconvulsive therapy is an important therapeutic option. This patient had received electroconvulsive therapy in

the past for the treatment of catatonia, and such therapy has a broad spectrum of effects for the treatment of multiple delusional conditions, including Cotard's syndrome.[27] Transcranial magnetic stimulation has had some promising effects in patients with catatonia.[33]

I suspect that this patient had neuroinflammation associated with Covid-19 that contributed to depression, catatonia, and Cotard's syndrome.

DR. GREGORY L. FRICCHIONE'S DIAGNOSIS

Cotard's syndrome, catatonia, and depression after coronavirus disease 2019.

DISEASE COURSE

Dr. Kritzer: After the patient arrived in the inpatient psychiatric unit of the other hospital, a nurse witnessed a generalized tonic–clonic seizure that lasted 30 seconds and was accompanied by urinary incontinence. Intravenous lorazepam and levetiracetam were administered, and the patient was transferred to the medical clinic of the other hospital for further care. The evaluation for precipitating causes of the seizure included a lumbar puncture for CSF analysis, including CSF testing for antibodies associated with autoimmune encephalitis, which was negative. The blood magnesium level was low, and there was evidence of a urinary tract infection. Additional imaging studies were obtained.

Dr. Paul: MRI of the head (Fig. 2) revealed no evidence of an acute infarct, intracranial mass, or hemorrhage. There was moderate confluent hypoattenuation involving the supratentorial white matter. The study did not show preferential involvement of the anterior temporal lobes and external capsules or show subcortical infarcts, findings that would suggest the diagnosis of CADASIL.[34] In addition, the study did not show restricted diffusion or microhemorrhages within the juxtacortical white matter, findings that would suggest the diagnosis of Covid-19–associated diffuse leukoencephalopathy and microhemorrhages.[35] There was no intracranial hemorrhage, which would suggest cerebral amyloid angiopathy.[36] There was mild generalized parenchymal volume loss that was advanced given the patient's age. The hippocampi were normal with respect to size, signal, and morphologic features. No cause of seizure was identified.

DISCUSSION OF SEIZURE MANAGEMENT

Dr. Zeina Chemali: An EEG recording (Fig. 3) revealed bilateral slowing without epileptiform discharges. There were several potential causes of a lowered seizure threshold in this patient, including treatment with bupropion, a urinary tract infection, and hypomagnesemia. Could Covid-19 have contributed to the seizure?

In a study that evaluated EEG recordings obtained from patients with Covid-19, nonspecific abnormalities of background rhythm were observed in most cases, with focal nonepileptic slowing found only around areas of other specific brain insults. Epileptiform discharges were observed in 20% of patients with Covid-19 who were in the intensive care unit, and nonconvulsive status epilepticus was diagnosed in 2.8% of these patients.[37] Potential mechanisms through which Covid-19 may contribute to seizures include direct viral invasion of the central nervous system (so far, this possibility has not been substantiated by research findings), exposure to glucocorticoids or other immunomodulatory treatments, or secondary effects of the illness, such as severe hypoxia, hyperthermia, thromboembolic events, or cytokine storm.[38] However, seizures occur in 150,000 people each year, and thus, the development of a seizure in this patient after the onset of Covid-19 could be a coincidence.[39] An international panel of experts recently determined that there is not enough evidence to suggest any direct correlation between Covid-19 and the potentiation of epileptic seizures.[40]

This patient was initially treated with levetiracetam, with a plan to administer a 6-week course followed by a tapering course over a period 1 to 2 weeks.[41] In addition, hypomagnesemia was corrected with the use of magnesium sulfate, the urinary tract infection was treated with nitrofurantoin, and the course of bupropion was tapered.

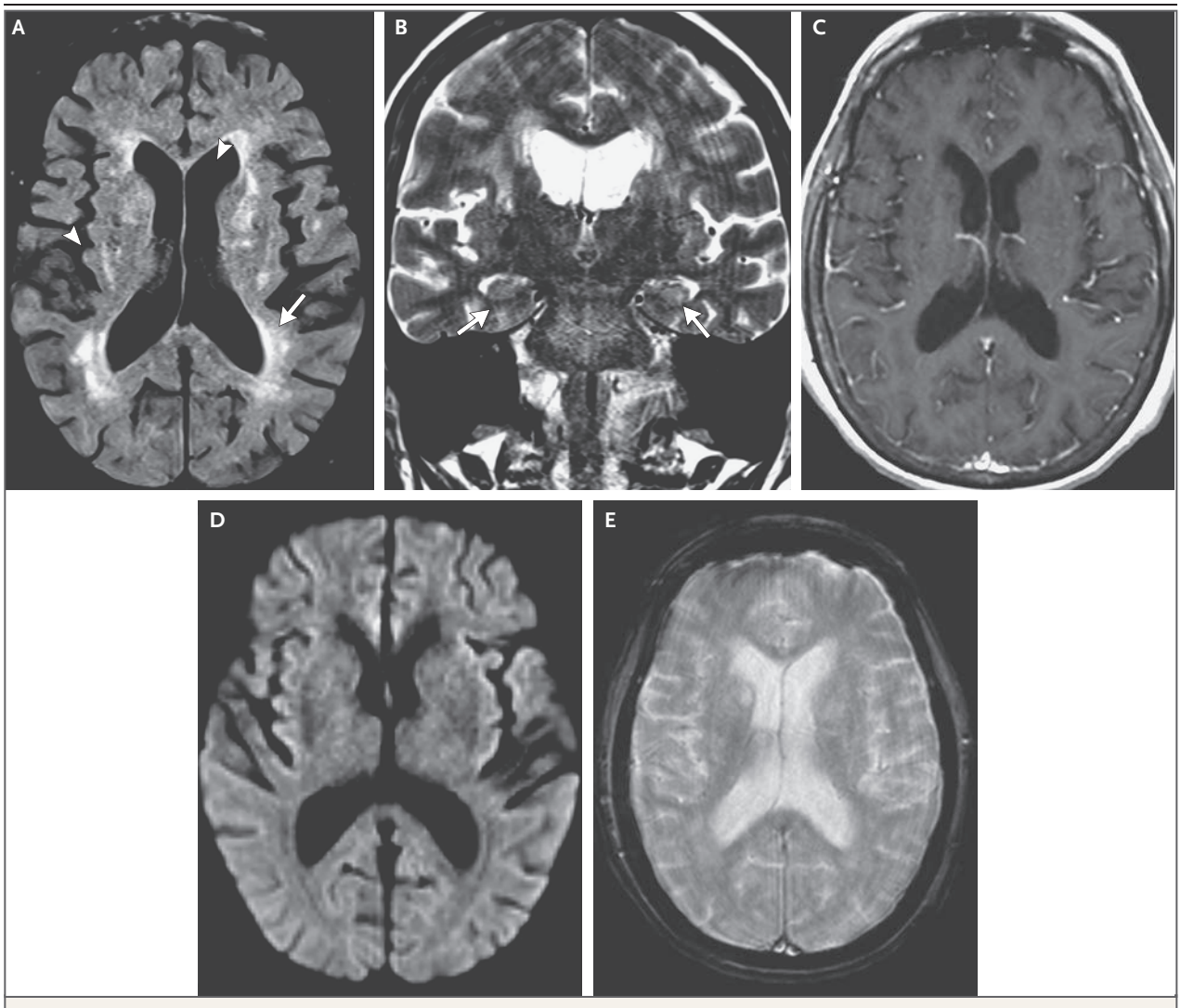

Figure 2. MRI of the Head.

An axial fluid-attenuated inversion recovery image (Panel A) shows moderate confluent hypoattenuation involving the supratentorial white matter (arrow), a finding consistent with small-vessel change, which is advanced given the patient's age. There is equivalent prominence of the ventricles and sulci (arrowheads), a finding consistent with mild generalized parenchymal volume loss, which is also advanced given the patient's age. A coronal T2-weighted image (Panel B) shows that the hippocampi are normal with respect to size, signal, and morphologic features (arrows). An axial T1-weighted image (Panel C), obtained after the administration of intravenous contrast material, shows no abnormal enhancement of the brain parenchyma. An axial diffusion-weighted image (Panel D) shows no restricted diffusion. An axial gradient–echo image (Panel E) shows no susceptibility signal. There is no evidence of a cause or consequence of seizure.

DISCUSSION OF PSYCHIATRIC MANAGEMENT

Dr. Kritzer: During the evaluation for precipitating causes of seizure, the patient had signs of delirium and catatonia. She had a score on the Bush–Francis Catatonia Rating Scale of 11 (with points for mutism, staring, verbigeration, rigidity, negativism, withdrawal, constant grasp reflex, autonomic instability, and repeatedly moving her arm in a circular manner). After one day of treatment with intravenous lorazepam, the score decreased to 5. Because the patient had mania and delirium, the dose of fluoxetine was decreased.

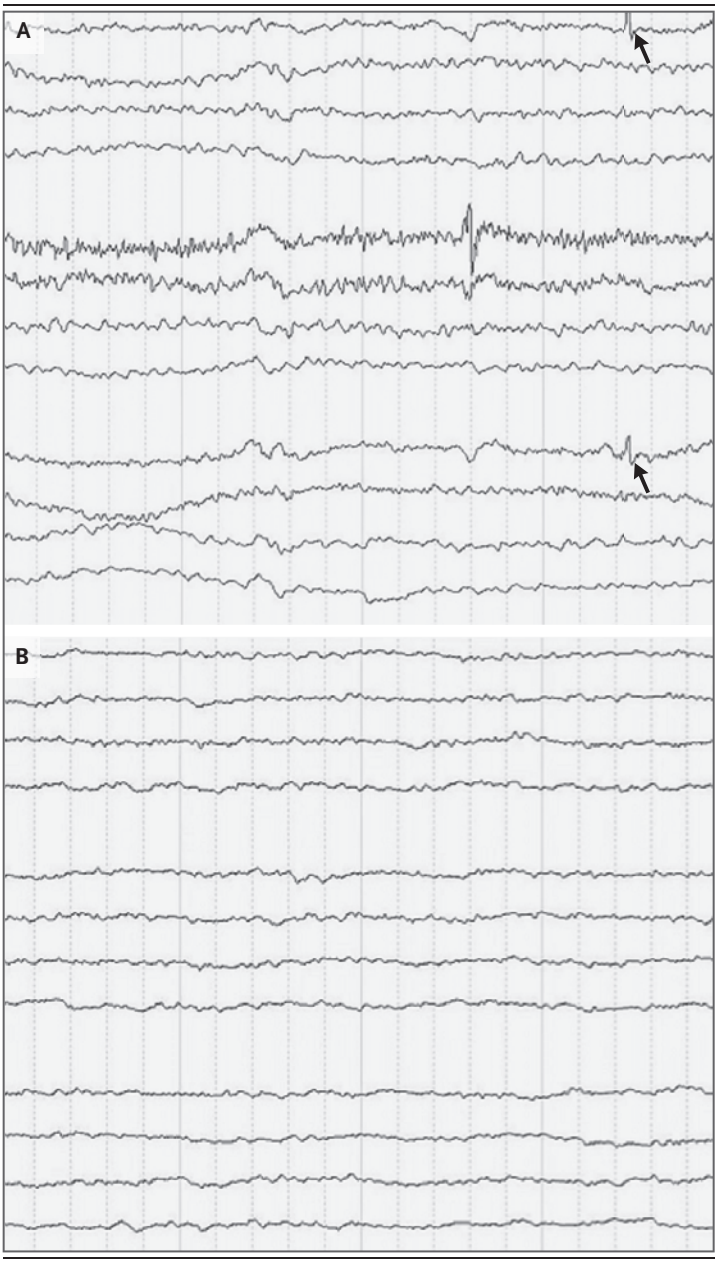

Figure 3. Electroencephalograms.

A bifrontal electroencephalographic (EEG) montage obtained after the patient had a seizure (Panel A) shows bilateral slowing without epileptiform discharges; there is a sharp frontal wave that may be an artifact (arrows). A follow-up EEG obtained 2 weeks later (Panel B) shows more slowing.

Once the patient's condition was considered to be medically stable, without overt signs of delirium, she was transferred back to the inpatient psychiatric unit. Electroconvulsive therapy was offered for the treatment of major depressive disorder and Cotard's syndrome, but the patient and her brother declined this treatment because they thought it had been ineffective in the past. There was ongoing agitation, and levetiracetam was switched to valproate to minimize neuropsychiatric side effects. The patient's condition improved during her monthlong hospitalization. The dose of lorazepam was gradually tapered, and the doses of valproate and olanzapine were adjusted.

Since discharge, the patient has been admitted to the inpatient psychiatric unit three times for major depressive disorder with psychotic features (mainly paranoia) or with poor self-care. She has had no recurrence of seizures and has reported a benefit from a recent trial of transcranial magnetic stimulation.

FINAL DIAGNOSIS

Cotard's syndrome, catatonia, depression, and seizure after coronavirus disease 2019.

This case was presented at Psychiatry Grand Rounds.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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